Matter is matter, neither noble nor vile, infinitely transformable, and its proximate origin is of no importance whatsoever.

Primo Levy, Italian chemist (1919–1987)

INDOOR AIR QUALITY

SHS Plus Ozone Poses One Fine Particle Problem

Smokers might want to think about creating a no-ozone zone as researchers report that the gas can react with chemicals in secondhand smoke (SHS) to produce ultrafine particles less than 100 nm in diameter.[1] "Given the very large surface area and the very high alveolar deposition fraction of such particles, their potential to cause health problems cannot be ignored," says first author Mohamad Sleiman, a chemist with the Environmental Energy Technologies Division of Lawrence Berkeley National Laboratory (LBNL).

SHS contains at least 250 known toxicants,[2] but until this study little was known about what is formed when SHS molecules react with ozone. Highly reactive ozone is a pervasive outdoor pollutant. It also is purposely produced indoors by certain air-purifying devices, ostensibly to remove airborne toxicants and odors like those from cigarette smoking (the actual effectiveness of these devices is debatable, however[3]). "We found that when the molecules in SHS react with ozone they can make ultrafine particles containing high-molecular-weight nitrogenated species," Sleiman explains.

The researchers generated SHS by letting 10 cigarettes smolder for 15 minutes in an environmental chamber of about the volume of an 8-×10-foot room. Then they pumped the contaminated air into 100-liter Tedlar® bags and added ozone-containing air to reach an initial ozone concentration of 110 ppb. The authors noted that the mass concentrations of both SHS and ozone used were representative of indoor environments where tobacco smoking occurred regularly and ozone air purifiers were in use.[1] The particulate matter in the resulting mixture was sized using a scanning mobility particle sizer, and its composition was examined using a time-of-flight mass spectrometer.

"What we found was surprising: large amounts of ultrafine particles, roughly eight times higher than those present in freshly emitted tobacco smoke," says coauthor Hugo Destaillats, also of LBNL. "Mass spectrometry showed these to be at least partly composed of high-molecular-weight nitrogenated oligomers that were not present in the original SHS." Indeed, he says, initial SHS compounds with a mass-to-charge ratio (a kind of molecular fingerprint) of less than 370 were much reduced in the postreaction sample while many new compounds with mass-to-charge ratios of around 400 to 500 had formed.[1]

Similar experiments performed with pure nicotine also produced ultrafine particles that contained some but not all of the same new compounds, showing that many of the oligomers had formed through reactions involving other components of SHS. However, the products of nicotine ozonolysis included many molecules with asthma hazard indices much higher than that of nicotine itself.[1] With a 4–9% total aerosol yield (the absolute aerosol mass) for the ozone–nicotine reactions alone, constant smoking could soon build up ultrafine particle concentrations in indoor air.

In recent years ultrafine particles have received increasingly bad press. Small enough to be inhaled deep into the lungs, where they can cross into the blood stream, they have been linked to a range of respiratory and cardiovascular problems through oxidative stress.[4] Indeed, ultrafine particles can enter cells themselves and even enter mitochondria, where oxidative stress is thought to damage the cristae.[5]

"The 'thirdhand' smoke products made by these reactions of SHS compounds with ozone would not just be inhaled," comments Jonathan Winickoff, an associate professor of pediatrics at Harvard Medical School. "After depositing on objects they could be absorbed through the skin or even ingested. Young children who explore the world by putting things in their mouths would be at greatest risk for this oral exposure route. When inhaled, these types of ultrafine particles place children at higher risk of asthma attacks." The full health implications of children's oral exposure are not yet well understood.[6]

A further problem would be the potential of these ultrafine particles to persist as residues on surfaces—perhaps for weeks—from which they could reenter the air over time, Sleiman says. "The reactants in smoke could also stick on surfaces, continuing to spawn ultrafine particles as they come into contact with ozone," adds Gary Cohen, a senior research scientist at the Karolinska Institute. "Smokers might therefore continue to poison the indoor environment, especially for infants and children, long after they have finished their cigarette."

Ozone air purifiers for commercial and residential use are sold to remove cigarette smoke toxics from indoor air and thus improve its quality. Ironically, the machines could be having exactly the opposite effect.

Joseph Tart/EHP

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HARMFUL ALGAL BLOOMS

Musty Warnings of Toxicity

On hot August days it's not uncommon for Midwestern swimmers and boaters to find their favorite freshwater beaches covered with musty scums of cyanobacteria, photosynthetic microbes formerly known as blue-green algae. Sometimes cyanobacteria produce toxins, an event called a harmful algal bloom (HAB). A recent survey of cyanobacterial blooms in 23 Midwestern lakes by the U.S. Geological Survey (USGS) suggests the odoriferous compounds that often accompany HABs may serve as sentinels of risk in recreational waters.[1]

Cyanobacteria produce a complex mixture of hepatotoxins and neurotoxins. Some species also produce relatively nontoxic "taste-and-odor" compounds such as geosmin and 2-methylisoborneol (MIB). These compounds, also found in soil and mushrooms, have strong earthy tastes and odors that people can detect at water concentrations of 10 ppt or less.[3]

In their preliminary study, Jennifer L. Graham and colleagues found geosmin and/or MIB co-occurred with cyanotoxins in about 91% of the blooms tested.[1] Because cyanotoxins occurred more frequently than geosmin and MIB, the authors cautioned that taste and odor can't provide surefire warnings of toxicity. However, a USGS press release on the study highlighted the need for increased cyanotoxin surveillance during taste-and-odor events so the public can be advised if necessary.[4]

The possibility of using taste-and-odor cues to help detect the presence of cyanotoxins is good news, because visual cues are notoriously equivocal. "A nasty-looking bloom doesn't necessarily mean a toxic bloom," says Mary Skopec, stream monitoring coordinator for the Iowa Department of Natural Resources. On the other hand, toxins may be left in the water after a bloom blows offshore or disperses, even if the water looks safe.

In the United States, recreation is the primary route of cyanotoxin exposure. Between 2005 and 2009 at least 19 states issued health advisories or closed recreational areas due to HABs, which can look like floating pools of blue, green, red, or brown paint.[5,6] Recreational exposure can cause gastroenteritis, rashes, asthmalike symptoms, abnormal liver function, weakness, and dizziness.[2] In countries where water treatment may be unreliable or unavailable, contaminated drinking water has caused serious disease and death in humans—for instance, exposure through drinking water has been linked to liver cancer in China.[2]

Children are at greater risk of recreational exposure than adults because "they tend to spend more time in the water and swallow more water," says NIEHS toxicologist Michelle J. Hooth. Livestock and pets also are more vulnerable, attracted by the same earthy smell that repels humans.[7] "Dogs can suffer seizures and die within minutes of coming out of [contaminated] water," Graham says. "It's very traumatic for a dog owner. People call us and want to know why they didn't know about the danger."

A boy fishes in Pawnee Lake near Lincoln, Nebraska, on 1 July 2005. HAB alerts were posted for Pawnee Lake for 14 weeks of the summer of 2005.

AP Photo/Nati Harnik

Angela Shambaugh, an aquatic biologist for the Vermont Department of Environmental Conservation, says concerns over two pet deaths in 1999 provided impetus for the initiation of cyanotoxin testing at Lake Champlain (which straddles New York, Vermont, and Quebec) and a successful communications program, including weekly online updates.[8] This summer, when "we had an expansive and very colorful cyanobacteria bloom on the main portion of the lake, most residents, though unfortunately not all, knew to keep children and pets out of the algae," Shambaugh says.

The U.S. Environmental Protection Agency has not set standards for cyanotoxin exposure for either recreational or drinking water, although it has added microcystin-LR and other cyanotoxins to its drinking water Contaminant Candidate List for further research.[9] "For most cyanotoxins we don't have enough toxicological data to come up with good guidelines," Graham says. Moreover, the environmental factors that trigger any given bloom to produce high levels of toxin are complex and difficult to predict.[2]

Cyanotoxins can be effectively removed from drinking water by a variety of treatment procedures.[2] But treatment is not foolproof, and in August 2010 microcystin-LR was detected in finished drinking water from three Ohio water systems, although none of the incidents were severe enough to warrant a drinking water advisory.[10] "The issue of cyanotoxins is on the radar for everyone that uses surface water," says Chris Jones, laboratory supervisor at Des Moines Water Works in Iowa, which draws drinking water from the Raccoon and Des Moines rivers.

The potential utility of taste and odor as a signal of toxicity is complicated by the fact that a musty smell doesn't necessarily mean treated tap water is unsafe. Treatments that successfully remove cyanotoxins may not eliminate geosmin and MIB,[11] which Jones says are "very water soluble." And if a HAB develops more quickly than the Des Moines Water Works can ramp up treatment with activated carbon, he says, "We can get calls from people saying the water tastes like dirt."[12]

But even though geosmin and MIB are not perfect indicators of presence of cyanotoxins, musty smells combined with the presence of cyanobacterial blooms can still serve as a "good warning tool" for recreational waters, says Keith Loftin, a coauthor of the USGS paper. "People can tell very quickly if something looks and smells bad."

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SMOKING AND SECONDHAND SMOKE

Study Finds No Level of SHS Exposure Free of Effects

How much exposure to tobacco smoke can the lungs endure before damage ensues? The answer appears to be none, based on gene activity measured by researchers at Cornell University.[1] “No level of smoking or exposure to secondhand smoke [SHS] is safe. Even at the lowest detectable levels of exposure, we could detect changes in gene expression within the cells lining the airways,” says coauthor Ronald Crystal, head of pulmonary and critical care medicine at New York-Presbyterian/Weill Cornell Medical Center.

Crystal and coworkers at Cornell analyzed gene activity in small airway epithelial cells collected from 121 healthy volunteers. The type of cells tested are where early damage first occurs that leads to chronic obstructive pulmonary disease (COPD) and bronchogenic cancer, according to Crystal.

The volunteers, all of whom had normal lung function, were categorized by tobacco smoke exposure status as determined by their urine levels of nicotine and cotinine. Nonsmokers had nondetectable urine nicotine or cotinine levels, low-exposure individuals had urine nicotine and/or cotinine levels up to 1,000 ng/mL, and active smokers had urine nicotine and/or cotinine levels greater than 1,000 ng/mL. The low-exposure group included occasional smokers and people exposed to SHS.

The researchers first compared the smokers and nonsmokers. Microarrays detected significant changes between these two groups in the activity of 372 genes. Among the low-exposure group, about a third of these 372 genes were up- or downregulated compared with nonsmokers, and 11% of the genes differed compared with active smokers.[1]

Even subjects with the lowest levels of nicotine and cotinine had enhanced activity of biological pathways involved in the metabolism of xenobiotics by cytochrome P450 and arachidonic acid. The same two pathways also were highly activated in smokers, suggesting exposure to low levels of SHS caused changes in the airways similar to those from active smoking, representing the earliest biologic abnormalities that can lead to disease.[1] The authors believe this may be the first study to document biological changes in the lung cells of people exposed to low levels of tobacco smoke.

The results support epidemiologic studies that link early respiratory damage to low levels of SHS exposure or occasional smoking.[2,3] However, the tobacco smoke–induced gene changes “do not tell us which ones [genes] are dangerous and which are protective,” Crystal notes.

Moreover, the cross-sectional nature of the study precluded determining whether the genetic changes predicted disease. Followup studies lasting 20 years or more are needed to sort out the genes that play a role in the development of lung diseases, and Crystal plans to follow some of the people in this study.

People often wonder what level of exposure to SHS is harmful—is it a problem, for instance, to hang out with smoking friends once or twice a week? Crystal’s study “employs sophisticated molecular genetic techniques to address this very important public health question of whether a threshold exists,” says Norman Edelman, a professor of preventive medicine at Stony Brook University Medical Center and chief medical officer at the American Lung Association. The finding that no level of tobacco smoke exposure appeared safe “is important for informing both individual behavior and public health policy,” Edelman says.

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The Beat

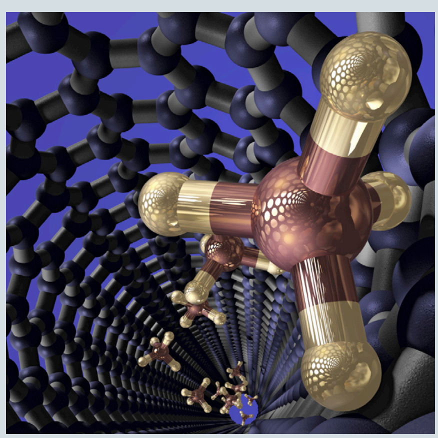
by Erin E. Dooley

EFSA on Revising BPA Guidance: Not Enough Evidence

In September 2010 the European Food Safety Authority (EFSA) released the findings of its latest review of bisphenol A (BPA), concluding there is no new evidence that warrants a revision of the current Tolerable Daily Intake of 0.05 mg/kg body weight.[1] EFSA also concluded that currently available animal data do not provide convincing evidence of neurobehavioral toxicity of BPA. The EFSA panel said it would reconsider the current opinion should new relevant data become available.

EPA Issues SNURs for Carbon Nanotubes

Significant new use rules went into effect 18 October 2010 for generic multi-walled carbon nanotubes and single-walled carbon nanotubes.[2] Carbon nanotubes currently are used in applications such as advanced composites, electronics, and fuel cells. Now companies that manufacture, import, or process these materials must notify the U.S. EPA 90 days before using them in a way that is deemed a significant new use. In May the GAO issued a report calling on the EPA to strengthen its oversight of nanomaterials used in commerce.[3]



Martin McCarthy/iStockphoto

PM Pollution: An App for That

University of Southern California researchers have developed a smartphone application to estimate atmospheric particulate matter.[4] The app currently works with Android systems, and an iPhone app is being developed. Users upload their photographs of the sky to a central computer, which compares the picture with established models of sky luminance to determine visibility, a measure associated with particulate pollution. The system then returns a message to the user and registers the information.

Updated Green Guides Open for Comment

In June 2010, *EHP* reported on the growing use of environmental stewardship claims in product marketing.[5] Now the Federal Trade Commission has issued proposed changes to its Green Guides, which aim to help marketers determine if their “green” claims are true and substantiated.[6] The Green Guides were last updated in 1998, well before a recent escalation in the

RESPIRATORY HEALTH

Measuring the Health Effects of Crop Burning

What to do with crop residue left in fields at the end of a growing season is, literally, a burning issue. Some farmers prefer the inexpensive approach of setting the stubble ablaze, but repeated burning is not good for the soil,[1] and the resulting smoke is a health hazard.[2] Although many studies have measured the particles released into the air by crop burning, fewer have isolated the effect of the smoke on lung function. New research now shows the smoke produced by crop burning could have a lasting effect on children's lung function.[3]

Ravinder Agarwal, head of the University Science Instrumentation Centre at Thapar University in Patiala, India, and colleagues used portable spirometers to regularly test the lung function of children aged 10–13 and adults aged 20–35 over the course of a year. The 40 participants were healthy nonsmokers living in a village surrounded by farmland, with little traffic and no industry within 10 km.[3]

Children's force vital capacity (FVC)[4] dropped from a mean 98% in August 2008 to 92% in July 2009. Mean FVC dipped as low as 88% in October and November, when farmers burned their rice crop residue, and in April and May, when they burned wheat stubble. The children's mean lung function remained significantly lower throughout the test period. The mean lung function of the adult study participants declined during the burn seasons as well, but largely returned to original levels by the end of the study.[3]

Decreases in lung function correlated with increases in the concentration of particulate matter, which exceeded India's national air quality standards during the burn season.[3] Small particles ($PM_{2.5}$ and PM_{10})—which make up the majority of the smoke produced by crop burning—were more closely associated with decreases in lung function than suspended particulate matter (SPM), which can contain particles 100 µm or larger.[5]

The findings linking seasonal burning with health issues "coincide with the anecdotal evidence that we have been seeing in the Canadian prairies," notes Kate Letkemann, environmental issues coordinator of The Lung Association, Manitoba, and a member of the provincial Crop Residue Burning Advisory Committee. On top of regulations regarding what time of day and where crop residue can be burned,[6] Manitoba uses incentives to encourage farmers to adopt alternative residue management practices, says Andrew Nadler, coordinator of the governmental Manitoba Crop Residue Burning Program. In the United States, crop burning is regulated at the state level.[7]

Argawal's work "builds a relationship between pulmonary function tests and the concentration of SPM, PM_{10}, and $PM_{2.5}$," notes Shijian Yang of the School of Environmental Science and Engineering at China's Shanghai Jiao Tong University. But he would like to see further research that looks closely at the dose–effect relationship between lung function and crop residue burning. Yang's work has shown that the peak concentration of PM_{10} and its duration may be more important than average concentrations for estimating the health effects of burning crops.[8]

Tina Adler first wrote for *EHP* about the Clinton–Gore environmental agenda in 1993. She is a member of the National Association of Science Writers and the American Society of Journalists and Authors.

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number of advertisements touting claims of environmental friendliness.[5] The proposed revisions include new guidance on the use of product certifications and other labeling tools. They also contain the first federal guidelines for the marketing of carbon offsets and renewable energy claims. The proposals are open for public comment until 10 December 2010.[7]

Left to right: Shutterstock; © Richard Martyniak/iStockphoto

Database of Bedbug Resources

A new online resource offered by the U.S. EPA aids consumers battling bedbug infestations.[8] The database lists about 300 pesticides that have been registered for use on bedbugs, and users can search for products that meet specific needs. The site emphasizes the importance of proper use of pesticides. The EPA Office of Pesticide Programs advises that pesticides work most effectively against bedbugs when used along with other steps such as reducing household clutter, using protective covers on mattresses, and vacuuming regularly. Bedbugs are classified by the U.S. EPA as "a pest of significant public health importance" under the Federal Insecticide, Fungicide, and Rodenticide Act.[9]

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Spent skins, eggs, and carcasses of the bedbug (*Cimex lectularius*).

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